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EFFECTS OF LONG DURATION TRAINING ON
HIGH DENSITY LIPOPROTEIN CHOLESTEROL AND RELATED
SERUM LIPIDS

By

Robert J. Confessore

B.S., Brooklyn College, City University of New York, 1977

Presented in partial fulfillment of the requirements

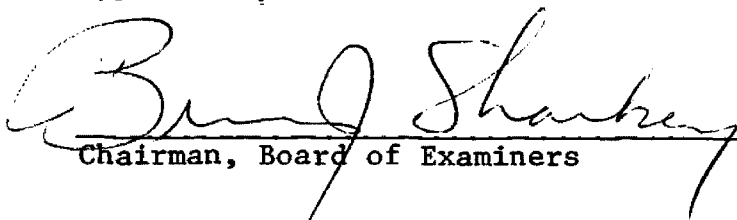
for the degree of

Master of Science

UNIVERSITY OF MONTANA

1979

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Dean, Graduate School

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Confessore, Robert J., Master of Science, September 17, 1979, Physical Education

Effects of Long Duration Training on High Density Lipoprotein Cholesterol and Related Serum Lipids

Director: Dr. Brian J. Sharkey 

This study investigated the effects of long duration training on the levels of HDL cholesterol and related serum lipids, in a sample of middle aged, relatively sedentary men and women. Volunteers from the University of Montana staff and faculty, and selected employees of the United States Forest Service were screened for participation in the study on criteria of age, maximum oxygen uptake, and percent body fat. Twenty-six subjects who met the criteria for participation in the study were placed into two training groups, according to their personal preference. The groups were designated: Group A (high compliance, long slow distance) and Group B (sports). A third Group C (low compliance, active control) was composed of subjects who did not fully comply with the training program. Subjects in the training groups engaged in twelve weeks of either long duration, low intensity jogging or basketball and racquetball. Exercise sessions were one hour in duration and were scheduled three days per week. Following the training period the pre and post training measurements were compared within each training group and between combined training groups.

The mean difference in HDL cholesterol for all subjects was not statistically significant. A highly significant difference in HDL cholesterol change existed between Group A and Groups B and C. The substantial increase in HDL cholesterol recorded in Group A was associated with a forty percent reduction in coronary heart disease risk. The initial level of HDL cholesterol was significantly correlated with the change in HDL cholesterol.

Groups A and C experienced increases in total cholesterol, while Group B showed a decrease in the total cholesterol level. A slight increase in the HDL/total cholesterol ratio was observed among exercising subjects. There was a significant increase in maximum oxygen uptake among the combined training groups, with a highly significant correlation existing between the change in maximum oxygen uptake and the weekly caloric expenditure (vigorous activity, > 7.5 cal/min). Overall reductions in body weight and percent body fat were observed, however these changes were not statistically significant.

It was concluded that low intensity, long duration exercise had a significant effect on the HDL cholesterol levels of those relatively sedentary, middle aged subjects, who complied with the twelve week training program. The initial level of HDL cholesterol is inversely related to the amount of change that can be expected as a result of exercise training.

ACKNOWLEDGEMENTS

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TABLE OF CONTENTS

	Page
ABSTRACT	ii
ACKNOWLEDGEMENTS	iii
LIST OF TABLES	vii
LIST OF FIGURES	viii
 Chapter	
1. INTRODUCTION	1
PURPOSE OF THE STUDY	4
DEFINITIONS	4
2. SURVEY OF RELATED LITERATURE	7
ATHEROSCLEROTIC DEVELOPMENT PROTEIN LIPID RELATIONSHIPS	7
HIGH DENSITY LIPOPROTEIN AND ATHEROSCLEROTIC DEVELOPMENT	13
EXERCISE AND CORONARY RISK FACTORS	17
EXERCISE AND TOTAL CHOLESTEROL/ TRIGLYCERIDE CONCENTRATIONS	19
EFFECT OF EXERCISE ON LIPOPROTEIN CONCENTRATIONS	25
3. METHODS AND PROCEDURES	29
SUBJECTS	29
INITIAL SCREENING	30
TRAINING	34
POST TESTING	35
4. ANALYSIS AND DISCUSSION OF RESULTS	36

Chapter	Page
COMPARISON OF CHANGES WITHIN TRAINING GROUPS	36
HDL CHOLESTEROL	36
TOTAL CHOLESTEROL	42
HDL CHOLESTEROL/TOTAL CHOLESTEROL RATIO	45
OXYGEN UPTAKE	46
CALORIC EXPENDITURE	47
PERCENTAGE BODY FAT	50
BODY WEIGHT	51
TRIGLYCERIDES	51
DISCUSSION	52
5. SUMMARY, CONCLUSIONS AND RECOMMENDATIONS	58
SUMMARY	58
CONCLUSIONS	59
RECOMMENDATIONS	60

APPENDIXES

Page

A. INFORMED CONSENT FORM	66
B. SKINFOLD NOMOGRAM	67
C. MODIFIED BALKE TREADMILL PROTOCOL	68
D. PERSONAL PERFORMANCE LOG	69
E. PROCEDURE FOR TOTAL CHOLESTEROL DETERMINATIONS	71
F. PROCEDURE FOR SEPARATION OF HIGH DENSITY LIPOPROTEIN FROM SERUM	72
G. CORONARY HEART DISEASE RISK TABLES	73
H. INDIVIDUAL DATA, GROUP A	74
I. INDIVIDUAL DATA, GROUP B	75
J. INDIVIDUAL DATA, GROUP C	76
K. PRE TEST PROCEDURES	77
L. INTERVIEW AND TESTING PROCEDURES	78
M. LIFESTYLE QUESTIONNAIRE	79

LIST OF TABLES

Table	Page
1. PHYSICAL CHARACTERISTICS OF SUBJECTS	33
2. EFFECTS OF TRAINING ON ALL EXERCISE GROUPS	38
3. EFFECTS OF TRAINING ON GROUP A (HIGH COMPLIANCE, LONG SLOW DISTANCE)	39
4. EFFECTS OF TRAINING ON GROUP B (SPORTS)	40
5. EFFECTS OF TRAINING ON GROUP C (LOW COMPLIANCE, ACTIVE CONTROL)	41

LIST OF FIGURES

Figure	Page
1. THE APPROXIMATE CHEMICAL COMPOSITION OF THE MAJOR LIPOPROTEINS	6

Chapter I

INTRODUCTION

Exercise has been proposed as a means of decreasing the risk of coronary heart disease, the current nemesis of modern society. The underlying disorder, atherosclerosis, accounts for approximately one half of all deaths in the western world (24).

Numerous epidemiological studies (12, 20, 24, 25, 32, 41, 52), have been conducted comparing the incidence of coronary heart disease and the amount of physical activity and working capacity. In a study released in November 1977, Dr. Ralph S. Paffenbarger Jr., of the Stanford University School of Medicine, showed that among 17,000 Harvard University alumni, examined during a fifteen year period, those who expended less than 2000 calories per week in exercise were 64% more likely to suffer heart attacks, than those engaged in strenuous activity (35).

Various predisposing factors associated with coronary heart disease have been reported to be favorably influenced by exercise. These factors include blood clotting and fibrinolysis, blood pressure, serum lipids, and myocardial function.

Development of coronary heart disease has been associated with a number of lipid risk factors. Elevation of triglycerides, total serum cholesterol and low density lipoprotein cholesterol (LDL), have been directly related to coronary heart disease prevalence (10).

Effect of exercise on total serum cholesterol has been the focus of a manifold of epidemiological and experimental studies. Mann et al. (32) reported a decrease in total cholesterol following a twelve week training regimen. Golding (19) and Rochelle (39) recorded reductions in total serum cholesterol after twenty-five and five week training programs respectively. Skinner and Holloszy (43) concluded from their studies that it is not impossible in some males to lower chronically elevated serum cholesterol. Campbell (7) recorded significant reductions in serum cholesterol following a ten week training period. These decreases were independent of weight changes and fluctuations in diet.

Rochelle (39), in reviewing early literature, provided possible mechanisms by which exercise may provide some degree of cardioprotection. One hypothesis stated that physical activity, by increasing metabolism, speeds up the process of cholesterol excretion, and also prevents synthesis of the sterol. Increased physical activity therefore resulted in a greater mobilization of alimentary assimilation. Related hypotheses maintained that cholesterol mobilization is enhanced by the massaging action of the muscles in clearing fats from the arterial walls, which in turn lowers the rate of fat deposition. Rochelle concluded that one can only postulate whether physical training produces its beneficial results by decreasing the blood cholesterol level, or by increasing collateral circulation and circulatory efficiency. He proposed that both may play important roles.

Cholesterol does not exist freely in the bloodstream, but is transported by macromolecules known as lipoproteins. Four major lipoproteins have been identified in human serum. These molecules vary in composition and density (figure 1). The total serum cholesterol measurement is the sum total of the cholesterol contained in very low density lipoprotein (VLDL), low density lipoproteins (LDL), high density lipoproteins (HDL), and chylomicrons.

Lopez (31) was perhaps the first to suggest that simultaneous changes in cholesterol among these fractions may conceal the effects of exercise. It was theorized that reductions in VLDL and LDL could be masked by an increase in HDL. Recent studies have reported elevated levels of HDL in groups exhibiting high levels of physical activity and concomitant below average incidence of coronary heart disease (1, 20, 24, 35, 38, 54).

Castelli (10), director of laboratories for the Framingham Heart Study, a case-control study encompassing five populations, reported an inverse association between coronary heart disease prevalence and levels of HDL. The strength of this association enabled Castelli to conclude that high density lipoproteins are "the single most powerful lipid predictor of inverse cardiovascular risk." Other major case studies have reported similar associations (20, 24, 36, 38).

These findings served as a stimulus for the initiation of several training studies investigating the effects of exercise on the high density lipoprotein fraction (29, 37, 40, 42, 47, 49, 50). The training programs of a majority of studies reporting positive results

have consisted of habitual levels of long duration, low intensity exercise (29, 47, 50, 54). Recent attempts have been made at quantifying the exercise in terms of intensity and duration in an effort to ascertain a training regimen that will provide the greatest possible positive effect (7, 49). The lack of conclusive evidence regarding exercise intensity and duration and HDL cholesterol suggested a need for further investigations.

PURPOSE OF THE STUDY

The purpose of this investigation was to determine the effects of long duration, low intensity exercise on HDL and related serum lipids. A second purpose was to compare these effects with those resulting from low levels of physical activity, and two popular sports.

DEFINITIONS

To facilitate the understanding of the remainder of this paper, terms that may be confusing or unfamiliar are defined.

ATHEROSCLEROSIS: form of arteriosclerosis in which there are localized accumulations of lipid-containing materials within or beneath the intimal surface of blood vessels. It is thought to be due to a metabolic defect in the smooth muscle cell involving lipids and lipoproteins, and is one of the most common causes of arterial occlusion.

CHOLESTEROL: is a sterol widely distributed in animal tissues. It can be synthesized by the liver and is a normal constituent of bile. It serves as a precursor of various steroid hormones, like sex hormones

and adrenal corticoids.

VERY LOW DENSITY LIPOPROTEINS (VLDL, pre-beta): small, light, glyceride rich (60-80%) lipoproteins that carry endogenous triglycerides, originating predominately in the liver, from the liver to sites in muscle and adipose tissue.

LOW DENSITY LIPOPROTEINS (LDL, beta): lipoprotein remnant partially or completely resulting from the metabolism of VLDL and carrying some of the protein, cholesterol and phospholipid of VLDL.

HIGH DENSITY LIPOPROTEINS (HDL, alpha): smallest of lipoproteins.

Their function is not completely understood.

CHYLOMICRONS: largest and lightest of lipoproteins, consisting of exogenous triglyceride.

TOTAL CHOLESTEROL MEASUREMENT: sum total of cholesterol contained in VLDL, LDL, HDL, and chylomicrons.

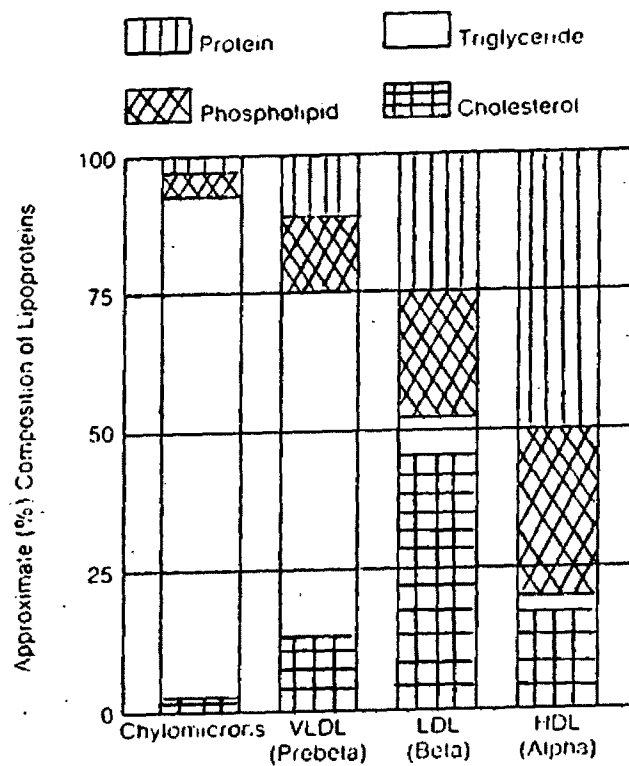


Figure 1

The Approximate Composition of the
Major Lipoproteins (54)

Chapter 2

REVIEW OF LITERATURE

ATHEROSCLEROTIC DEVELOPMENT: PROTEIN LIPID RELATIONSHIPS

Atherosclerosis is a complex process which may be regarded as a dynamic interaction among: (1) the structural and metabolic properties of the arterial wall, (2) the components of the blood, and (3) the hemodynamic forces. (17)

The artery wall is composed of three layers: the intima, the media, and the adventitia. Lining the inner surface of the intima is a single layer of endothelial cells, which hold the blood cells within the artery and modulate the passage of water and other substances from the blood plasma into the tissues. It is in the intima that atherosclerosis has its effect. The characteristic lesions of atherosclerosis are raised fibrous plaques. The main cellular component of the plaque is the smooth muscle cell, very similar to the major cell of the normal artery wall. Macrophages and other white blood cells infiltrate the plaques' dense connective tissue which consist largely of collagen fibers. The plaque usually contains glycoaminoglycans, and sometimes elastin, and such blood proteins as fibrinogen. Lipoproteins are found both inside and outside cells. Deep within the lesions there are debris from dead and dying cells, and varying amounts of lipids. Crystals of cholesterol can sometimes be seen,

even with the unaided eye, in the softened debris of advanced lesions. It is this fatty debris that suggested the name atherosclerosis, from the Greek, athera (gruel) and sclerosis (hardening).

The origin of atherosclerosis to this day remains a mystery. Numerous theories and hypotheses have been put forth in attempts at explanation. The major theories have been reviewed by Benditt (3).

The insudation or infiltration theory contends that infiltration of fatty substances from the bloodstream into the arterial wall gives rise to deposits of cholesterol that act as an irritant, causing inflammation and the proliferation of cells. A long chain of investigation establishes beyond doubt the importance of lipids as constituents of the atherosclerotic lesions. It also establishes cholesterol as the most important lipid, quantitatively, but is only suggestive as far as the origin of the lipids in the lesions is concerned, and is even less informative about the primary event responsible for the deposition of the lipids (15).

The encrustation theory suggests that plaque originates as a small mural thrombus. This mural thrombus is then converted into a mass of tissue in the intima, as arterial wall cells migrate to it, multiply, and secrete the characteristic extra cellular substances.

Ideas about the nature and origin of atherosclerosis began to change when the electron microscope revealed the cellular composition of plaques. The fibrous cellular cap covering the cholesterol rich debris turned out not to contain ordinary fibroblasts, the cells that proliferate to heal a skin wound, but rather than to contain smooth

muscle cells, similar to those of the normal artery wall. It was then discovered that in its early stages, the human plaque does not contain much lipid, which implied that lipid insudation is not the primary initiating factor.

The major current theory of the genesis of atherosclerosis is known as the monoclonal hypothesis. This hypothesis holds that the proliferating cells of an atherosclerotic plaque all stem from one mutated cell. Related theories share the common belief that the lesions begin as localized excessive accumulations of smooth muscle cells in the intima.

Benditt (3) describes at least two major stages in the atherosclerotic process:

Initiation Stage: precipitated by a mutation in artery cell wall. Factors and conditions then promote the expression of the selective proliferation advantage conferred by mutation, enlarging the mass of cells. The mutation may never be expressed as gross plaque formation unless something promotes cell multiplication and gives altered cells an opportunity to express their altered capability for growth.

Stage of Complication: tendency of cells to degenerate and lesions composed of those cells to breakdown and ulcerate is compatible with the presence of an altered cell population.

Getz (17) et al. list numerous critical factors, which presumably control the advancement of the disease:

1. Increase in quantity of beta or pre beta lipoproteins
2. Change in quality of lipid or lipoprotein
3. Decrease in stability of lipoproteins
4. Increase in clotting tendency or platelet sticking
5. Increased permeability due to hypertension
6. Plugging fragmentation or duplication of internal

- elastic membrane
- 7. Decreased metabolism of smooth muscle cells
- 8. Proliferation of smooth muscle cells
- 9. Poorly metabolized lipoproteins accumulating in smooth muscle cells
- 10. Irritating or toxic lipoproteins in smooth muscle cells leading to necrosis
- 11. Interruption of lymphatic drainage.

Blood lipoproteins seem to be good nutrients for culturing smooth muscle cells from the human artery wall. Several types of arterial injury, including those due to hypertension, appear to increase endothelial permeability to plasma macromolecules, including low density lipoproteins and blood cells, and thereby augment atherosclerosis (17).

Artery wall cells are capable of converting into mutagens certain pre-mutagenic substances that come from the environment and are transported to the cells by lipoproteins (3). Studies reviewed by Wissler (51) indicated that smooth muscle cells in atherosclerotic lesions seemed to have fewer mitochondria suggesting that once the smooth muscle cells accumulate in the intimal lesions their function also changes. There is also evidence that medial cells migrate into the intima. Several studies have been reported in which observers have witnessed smooth muscle cells actually lying within the fenestrae of the internal elastic membrane of the rabbit artery (51). Researchers speculated that the smooth muscle cells migrate from the inner media to the intima or vice versa. Wissler (51) concluded that in human atherogenesis there is a delicate balance among three more or less distinct relations:

1. Proliferation of myointimal cells to produce a

- thickened intima
- 2. Migration of medial cells into the intima
- 3. Alteration of the metabolic capacity of the myointimal cell, including definite cell injury or death.

Benditt (3) suggested some possible initiating factors in the development of atherosclerosis. These include intrinsic genetic factors, chemical mutations, viruses, and ionizing radiation. Although there is still much investigation to be done, the primary value of the monoclonal hypothesis is that it provides a new framework within which one can ask questions about the role of proposed risk factors such as cigarette smoking, dietary habits, changes in blood lipids, and hypertension.

All of the cholesterol and phospholipid of normal plasma is combined with protein and in combination either with high density lipoproteins (HDL), low density lipoproteins (LDL), very low density lipoproteins (VLDL), or chylomicrons. The composition of these macromolecules is shown in figure 1. Forty percent of the total serum cholesterol is derived from the diet, while the remainder is synthesized by the body, primarily in the liver and intestine. Homeostatic mechanisms are responsible for the control of the serum cholesterol level. The liver plays the most important role in the control of the serum cholesterol level, through its ability to synthesize cholesterol and form bile acids which serve to catabolize cholesterol (11)_λ. The bile acids are reused, but are eventually modified by bacterial action in the intestine and excreted in the feces. Mechanisms are also available to control the absorbtion of dietary cholesterol.

In discussions concerning atherosclerotic development, most attention has been focused on the cholesterol rich VLDL and LDL fractions. The availability of blood samples drawn from two patients during the weeks preceding death, and aortic tissue taken at necropsy made possible a direct study of the relationship between serum lipid levels and the concentration of intact lipoprotein in the aortic intima. Smith and Slater (44) suggest that a rather constant amount of whole plasma may be entering the intima, carrying a variable amount of lipoprotein dependent on its cholesterol level. The authors reported about seven times as much lipoprotein as albumin, which strongly suggests some form of preferential retention of the lipoprotein. Extracellular cholesterol esters are derived directly from plasma low density lipoproteins. LDL has been demonstrated in the arterial wall, and the authors found that the concentration of the lipoprotein was significantly higher in normal intima from men with myocardial infarction, than in men with other conditions. The authors found that the absolute amount of intimal lipoprotein increases linearly with serum cholesterol, and results suggested that the total amount of plasma entering the intima may increase with increasing blood pressure, so the effect of the two factors would be additive. The study has made clear that within the intima there is a substantial pool of soluble intact lipoprotein which increases with increasing serum cholesterol level, and which might become irreversibly bound or precipitated if metabolic changes occurred in the surrounding tissue.

HIGH DENSITY LIPOPROTEIN AND ATHEROSCLEROTIC DEVELOPMENT

A vast amount of evidence is beginning to accumulate pointing to an important function of the HDL fraction, in the slowing of the atherosclerotic process.

Barr (2), as early as 1951, observed that patients who survived coronary occlusion or present otherwise unequivocal evidence of the complications of atherosclerosis frequently exhibit several abnormalities in the distribution of proteins and lipids in the plasma. These included a tendency to reduction of albumin and alpha lipoproteins (HDL), and a relative and absolute increase in beta (LDL) lipoproteins.

Recent epidemiological evidence report similar observations. Castelli, et al. (10) studied the relationship between coronary heart disease prevalence and fasting lipid levels. This was assessed by a case-control study encompassing five populations. In each major study group mean levels of HDL cholesterol were lower in individuals with coronary heart disease, than in those without the disease. An inverse association was reported between HDL cholesterol and prevalence of coronary heart disease, and this association was not appreciably diminished when adjusted for levels of LDL cholesterol and triglyceride. HDL and LDL emerged as significant factors in coronary heart disease prevalence, whereas triglyceride was less consistently a significant factor.

In a population study of Hawaiian men, Rhoads (38), reported the inverse relationship of HDL to prevalence of coronary heart

disease, and found that this relationship was independent of LDL, obesity and other factors. It was also reported that prevalence rates of coronary heart disease rise with increasing levels of total cholesterol and LDL.

Findings of Rhoads et al. (38) have been substantiated by the work of the Framingham Heart Study. Results of that study enabled Dr. W. P. Castelli (24) to state that high density lipoproteins are the "single most powerful lipid predictor of inverse cardiovascular risk."

Bang, Dyerburg et al. (1) compared the plasma lipid and lipoprotein concentrations of Greenlandic West Coast Eskimos to levels of the same components in a Danish population, including Greenlandic Eskimos living in Denmark. Significantly lower values were found in the Eskimos for total lipids, cholesterol, triglyceride, VLDL and LDL when compared with age and sex matched Danish controls. HDL was generally higher in male Eskimos than in their counterparts. Median values for plasma total lipids were all significantly higher in Eskimos living in Denmark than in Eskimos living in Greenland. Examination points strongly against a genetic and towards an environmental explanation of the difference in lipid and lipoprotein concentrations.

Berg et al. (4) determined serum HDL concentrations in 49 subjects who had a myocardial infarction and in 102 healthy middle aged men from northern Sweden. The mean HDL concentration was found to be significantly lower in the men with coronary heart disease, than in the controls.

A recent study by Glueck (18) examined families with a history of longevity whose members reach their eighties and nineties in good health without serious cardiovascular disease. An unusually high level of HDL cholesterol was characteristic of these subjects. Levels of more than 75 mg/dl were reported.

Recent studies have proposed numerous mechanisms of HDL action. Low plasma concentrations of HDL may accelerate the development of coronary atherosclerosis by impairing the normal clearance of cholesterol from the arterial wall, and its transport to the liver for metabolism and excretion. This mechanism has been suggested by the studies of Miller and Miller (34). These researchers found that the body cholesterol pool, measured indirectly by isotope dilution or directly by chemical analysis, increases with decreasing plasma levels of HDL, but is unrelated to the plasma concentrations of total cholesterol and other lipoproteins.

Follow-up studies which have demonstrated that the invitro efflux of cholesterol from atheromatous tissue is promoted by the addition of HDL to the incubation medium, provide additional support for this theory which views HDL as a "scavenger" lipoprotein. It has been further proposed by Miller and Miller (34) that the development of atherosclerosis might be more successfully prevented by increasing plasma HDL, and hence the clearance of cholesterol from the arterial wall, then by conventional attempts to reduce plasma cholesterol and other lipoproteins.

HDL may facilitate the action of lecithin/cholesterol acyltransferase (LCAT), the enzyme that converts free cholesterol to esterified

cholesterol. HDL and LCAT do not work well without each other. Esterified cholesterol molecules are larger than free cholesterol molecules and move more slowly. As a result they have trouble passing back and forth across cell membranes. Thus, if cholesterol at the peripheral cellular level is held out of the cell for a time by LCAT action, there is a greater chance that it will be picked up by an HDL molecule and transported to the liver for elimination.

In a study of the metabolism of lipoproteins by arterial smooth muscle cells Carew et al. (8) demonstrated that HDL competitively inhibits the uptake and degradation of low density lipoproteins by the cells. This insures that the arterial smooth muscle cells accumulate less cholesterol. HDL binds to the surface of the muscle cells as effectively as LDL, but is internalized and degraded more slowly, significantly modifying the metabolism of LDL by these cells.

Koshinsky et al. (27) further explored the influence of HDL on binding, uptake, and degradation of LDL by non confluent pig smooth muscle cells. In cells incubated at 37°C with 125 I-LDL, after a 20 hour pre-incubation in lipoprotein deficient serum, uptake and degradation were both inhibited by HDL 57 and 48% respectively. These results together with previous observations suggest that LDL binding sites on the smooth muscle cells also interact to some degree with HDL. Incubation of human serum with crystalline cholesterol which had been pulverized by sonication resulted in a measurable uptake of cholesterol by the serum. Hiss et al. (21) designated this uptake "serum cholesterol binding reserve"(SCBR). SCBR could be attributed to two serum lipoprotein subfractions; SFV,

separated from VLDL and SFH from HDL by gel filtration. It is proposed that SFV and SFH have physiological roles in retarding atherogenesis by removing cholesterol from the arterial intima and transporting it back to the circulating serum. Accordingly, individuals who have low SCBR values, being deficient in SFH and SFV, are at a higher risk for the development of atherosclerosis and coronary heart disease. This hypothesis was tested by comparing SCBR values of patients with premature myocardial infarction, to values of controls. The results indicated a trend of increasing SCBR values with increasing levels of serum cholesterol; and triglycerides among the controls, but this trend was virtually lost among the patients. The SCBR values were also lower among patients than controls. The combined amounts of cholesterol solubilized by SFV and SFH, account for approximately 80% of the measured SCBR. Since SFV and SFH are constituents of normal serum and can specifically solubilize additional cholesterol, they are likely candidates to function as vehicles for reverse cholesterol transport.

Studies by Carew (8), Hisa (21), and Koschinsky (27) were conducted in an artificial environment, and some degree of caution is advised in the interpretation of these studies. In vivo studies are needed to confirm these results.

EXERCISE AND CORONARY RISK FACTORS

Numerous studies have explored the role of physical activity as a possible factor in the prevention of atherosclerosis and coronary heart disease.

Wolffe (52) surveyed three hundred athletes actively engaged in physical activity and three hundred executives, all past middle life, for evidence of atherosclerosis. Definite or suggestive evidence of atherosclerosis was found in a high number of the executive group, whose physical activities were curtailed. The athletes showed little evidence of atherosclerosis.

Cooper (12) constructed five age adjusted cardiorespiratory fitness categories, determined from treadmill data. He reported a consistent inverse relationship among physical fitness categories and resting heart rate, bodyweight, percent body fat, serum levels of cholesterol, triglycerides, glucose and systolic blood pressure. These results implied that physical fitness is related to lower coronary risk factors. Differences in coronary heart disease risk were minimal for adjacent levels of fitness groups, but became more marked among groups with greater differences in levels of fitness.

Kannel (25), reporting on findings at Framingham, stated that in the ten years following physical activity assessment, 207 men developed some manifestation of coronary heart disease. Those classified most sedentary in each age group had a coronary incidence rate almost twice that of those who were at least moderately active. Only among individuals dying of cardiovascular causes was the reported level of physical activity less than average. Framingham data strongly suggests that it is indeed the physical activity and not some coincidently associated coronary precursor which is responsible.

Hernsburg (20) studied 1,012 leading businessmen from different parts of Finland, who underwent extensive medical examinations. Hernsburg reported that the larger the physical working capacity, the lower the serum cholesterol level. Since all subjects were from the same occupational and socioeconomic group, the author concluded that the relationship between physical working capacity and serum cholesterol is not to any significant degree dependent on the nutritive state.

Paffenbarger (36) conducted a 15 year follow up study of 3,263 longshoremen. Men with more sedentary jobs expended 925 fewer calories per workday and sustained coronary death rates one-third higher than those among cargo handlers, a more active occupation. Rate differences were largest at youngest ages and decreased steadily thereafter. Paffenbarger suggested that physical activity may have a more striking influence on myocardial infarction than on atherosclerosis.

In a recent and much publicized study released in November of 1977, Paffenbarger (35) showed that among 17,000 Harvard University alumni examined during a 15 year period, those who expended less than 2,000 calories per week in exercise were 64% more likely to suffer heart attacks, than those engaged in strenuous exercise.

EXERCISE AND TOTAL CHOLESTEROL/TRIGLYCERIDE CONCENTRATIONS

Studies have indicated that the distribution of cholesterol among the lipoprotein classes may have effects on the atherogenic properties of cholesterol, and that the total serum cholesterol may play a role in the initiation or growth of the atherosclerotic lesion. It

is therefore vital to consider any factor, such as exercise, which may have favorable effects on these parameters. The majority of studies concerning the effects of exercise on serum cholesterol have dealt with its effects on the total cholesterol measurement.

Golding (19) found that total serum cholesterol levels were significantly reduced following a twenty-five week training period consisting of hard and severe all out endurance type activities. Training sessions were one hour in duration, five days per week. Golding found the drop in cholesterol values to be inversely related to attendance. The author further postulated that exercise may have increased liver function, causing the liver to empty cholesterol into the blood stream.

Rochelle (39) charted blood plasma cholesterol levels in six experimental and six control subjects, during a five week training program. The training consisted of a two mile run for time, five days per week, and also included an eight week de-training period. Plasma cholesterol levels were significantly reduced during the course of the intensive training. Plasma cholesterol levels returned to pre-training levels within four weeks after the cessation of training.

Some researchers involved in the area of exercise and its effect on the total cholesterol measurement felt it necessary to quantify the exercise in terms of type, intensity and duration.

Campbell (7) randomly selected 133 young adult males to participate in ten week programs of cross-country running, golf, tennis, tumbling and gymnastics, wrestling and weight training. Findings

suggest that different types of physical activity influence cholesterol levels in different degrees. Subjects who participated in a vigorous, dynamic type of activity showed a significant decrease in serum cholesterol, whereas subjects who participated in a vigorous but static type of activity experienced no significant reduction during the experimental period. Campbell defined dynamic activity as a rapid interchange in the position of the arms and legs, and rapid flexing and relaxation of muscles.

Skinner, Holloszy et al. (43) devised two experiments to explore the effect of exercise on serum cholesterol. For both experiments subjects were males who began the control period in a sedentary state. The initial control period was followed by three periods of exercise and subsequent control periods. Blood samples were drawn daily for the last five days of each period.

In the first experiment, exercise of calculated caloric quantity and intensity was measured on the treadmill. The quantities of exercise taken for at least 17 days per period, and at least six days per week were 300, 500, 900 Kcal, and the intensities were 500, 900, 1,000 Kcal/hr. The experiment lasted eight months. The serum cholesterol levels of the first and the last control periods were significantly higher than those associated with all levels of exercise. These results did not agree with experiments in which larger quantities of exercise were given at low intensity. The authors hypothesized that the intensity of the exercise was relatively more important than the duration.

In their second experiment the authors used exercise of constant quantity but varying intensity. As caloric cost decreased with training at the middle and high levels of exercise, work intensity was increased as required. The data suggest that sedentary men, with serum cholesterol levels of over 200 mg/dl, taking frequent high intensity exercise of 11-14 Kcal/Kg/hr, in quantities of 3 Kcal/Kg or more, will significantly lower serum cholesterol if diet is controlled. The authors conclude that it is not impossible to lower chronically elevated serum cholesterol through exercise.

Dressendorfer et al. (13) set out to determine if serum cholesterol and triglycerides were related to the amount of training. Eighty experienced male runners, and 64 male non-runners aged 22-59 years volunteered for the program. To be classified as a runner the subject must have averaged at least six miles of running in three or more training sessions per week. The intensity of the workout was not a classification factor. Runners were divided into three subgroups, low mileage 12 miles per week, moderate mileage 15-24 miles per week, and high mileage, over 28 miles per week. Each subgroup averaged more than four years of running at the weekly mileage. Results showed significantly lower total plasma cholesterol and triglyceride in runners compared to sedentary controls. There was no significant difference in serum cholesterol or triglyceride levels between the low mileage and high mileage subgroups of runners. Low mileage runners had cholesterol values similar to those of marathon runners, although their weekly mileage was only one-fifth as great.

Miles (33) paired 22 men aged 28-54, by age, and randomly assigned within each pair to either an experimental or control group. Training sessions were 45 minutes in duration and subjects were required to attend four times per week. The main body of the workouts included progressively harder bouts of interval training, continuous distance running, endurance calisthenics, bench stepping and eighty-five yard sprints. The duration of the training regimen was eleven weeks. Serum cholesterol was significantly reduced in the experimental group. Triglycerides increased 12.6 mg% in the experimental group, and 30.0 mg% in the control group.

Johnson, Wong et al. (23) studied healthy young varsity swimmers over a period of fourteen months with regard to the effect of a typical training and competitive collegiate swimming program on plasma cholesterol and phospholipids. The exercise used for conditioning varsity swimming athletes did not significantly lower serum cholesterol and phospholipids. However the authors determined that several factors seemed to influence the outcome. Such factors included very low initial lipid levels, interruptions in the training program, and modest caloric intake and maintenance of weight control.

Hurtler (22) measured the plasma concentration of unesterified fatty acid, triglyceride, cholesterol, phospholipids, and the relative amounts of the common fatty acids contained within these lipid fractions in fourteen trained athletes at rest and after a marathon race. The total cholesterol, triglyceride and phospholipid concentrations showed no significant change, but in each of these fractions there was a

relative reduction in unsaturated acid, and a corresponding rise in saturated acid, probably associated with preferential muscular oxidation of the unsaturated acid. When these runners were resting their plasma lipids were compared with those of fourteen sedentary men matched for age and physique. The mean plasma triglyceride level for the athletes was significantly lower than the controls, there was no significant difference between the plasma cholesterol or phospholipid concentrations of the two groups.

Several studies have examined the effect of exercise on plasma lipoproteins as well as total cholesterol and other plasma lipids.

It was the aim of Ratliff et al. (37) to determine the effects of chronic exercise training on plasma levels of triglyceride, cholesterol and fractions of HDL, LDL, and VLDL cholesterol. A group of previously untrained early middle aged firefighters engaged in an individually prescribed jogging program of progressing intensity and duration, three times per week for twenty weeks. Another group of firefighters served as inactive controls. Decreases were observed in triglycerides, cholesterol, LDL and VLDL in the exercise group. Only increases in HDL, maximal oxygen uptake and a decrease in percent body fat were found to be significant at the 0.5 level from the control group.

Roundy (40) studied the effects of ten weeks of supervised exercise on the same lipid measures. Subjects included fifty-one sedentary adults, with normal lipid patterns and with type II and IV hyperlipoproteinemia. Maintaining pulse rates at predetermined levels,

subjects engaged in continuous walking and jogging, three times per week. The length of the exercise periods was gradually increased. Exercise proved to be effective in relieving abnormal serum lipid conditions in subjects with type II and IV hyperlipoproteinemia, and in causing a favorable shift in lipoprotein densities from lighter densities (VLDL) (LDL) to high densities (HDL).

Leon et al. (29) observed six obese sedentary young college men on unrestricted diets. The subjects walked one and a half hours, five days per week on a treadmill (3.2 mph, 10% grade) for sixteen weeks. An average weight loss of 5.7 kg was associated with a 30% decrease in body fat. A reduction in mean levels of total plasma cholesterol, triglycerides, VLDL and LDL was reported, in addition to an increase in HDL.

Vial et al. (47) conducted a seven week exercise program involving four adult males. The program consisted of a daily thirty minute exercise period involving running, bicycling, calisthenics and jogging. A significant reduction in serum cholesterol, beta cholesterol and triglycerides was reported. Serum electrophoresis revealed a decrease in LDL and VLDL with an increase in the HDL fraction. Changes were also observed in LCAT enzyme activity. Weight remained unchanged, with a decrease in resting pulse rate.

Carlson and Mossfeldt (9) gathered data regarding changes in plasma lipids and lipoproteins during participation in ski racing over an 8-9 hour period. The race covered 85 Km over hilly terrain. All subjects had participated in a training program five months prior to

the race. Fasting levels for plasma concentration of cholesterol, phospholipids, and triglycerides were the same during skiing and during ordinary activities. During skiing there was a significant fall in all plasma lipids, while no significant changes occurred during normal activities. The key finding in this study was that the concentration of triglycerides and phospholipids in plasma was reduced during prolonged heavy exercise. Indications are that the changes observed during the race were due to the exercise and not to the carbohydrate load.

Lehtonen et al. (28) concluded that physical training produced a significant decrease in plasma cholesterol. The training consisted of a two mile run for time, five days per week for five weeks. A significant temporary rise in cholesterol occurs during exercise, possibly noting fat mobilization. The plasma cholesterol of each of the subjects returned to pre-training levels within four to six weeks after training was stopped. An increase in HDL was also reported. The authors further reported an inverse relationship between VLDL triglycerides and HDL, so that the decrease in serum triglycerides and the increase in HDL may reflect the same phenomenon.

EFFECT OF EXERCISE ON LIPOPROTEIN CONCENTRATIONS

In light of evidence regarding the distribution of cholesterol among the lipoprotein classes, and its relation to the atherogenic potential, recent researchers have focused specifically on the effects of exercise on lipoprotein levels.

Webster (50) studied thirty middle-aged men over a twelve week period. Fifteen subjects participated in forty-five minutes of jogging, three days per week, while remaining subjects served as controls. Following the first three weeks of training the trend was for the joggers to have higher levels of HDL cholesterol than the controls. Webster reported that these changes were independent of weight loss, adipose tissue loss, and diet.

Wood et al. (54) compared fasting plasma lipid and lipoprotein levels in forty-one male long distance runners aged thirty-five to fifty-nine years, with data for 890 males of similar age range randomly selected from three northern California towns. All runners averaged at least fifteen miles per week during the previous year and were in active training. The authors reported lower cholesterol, triglyceride, and LDL concentrations, and high HDL and HDL/LDL ratios for the runners, as opposed to the controls.

Washburn (49) investigated the effects of intensity and duration of training on HDL cholesterol. Exercise prescriptions were individualized for each of ten training subjects. Subjects were then assigned to two training groups: low intensity, long duration or high intensity short duration exercise. Analysis of data indicated that there were no significant changes within either exercise group in HDL cholesterol, total cholesterol, weight, percent body fat or blood pressure. Washburn further reported that negative correlations were found between initial level of HDL cholesterol and change in HDL cholesterol, and initial level of maximum oxygen uptake and change in maximum oxygen uptake.

Subjects in this study did have high initial fitness levels.

Simpson (42) reported that HDL cholesterol levels showed a highly significant positive association with intense physical activity (> 7.5 cal/min), but not with total activity. Positive changes in HDL levels were demonstrated at 2,000 Kcal per week of intense activity.

Chapter 3

METHODS AND PROCEDURES

SUBJECTS

Notice and information regarding project lifestyle was sent to all academic departments of the University of Montana and selected employees of the United States Forest Service, from a summer program conducted by Dr. Brian Sharkey. Notice of project lifestyle was sent to those Forest Service employees whose health risk analysis indicated that they might reduce their risk through participation in an exercise program. Notices distributed to the university community called for sedentary individuals between the ages of 25-45 years. Twenty-six male and female volunteers, who met the criteria for participation in the study, were selected as subjects.

Three training groups were identified according to the type of activity, and the level of compliance and participation of each individual subject. Assignment to training groups A and B were made according to the subjects personal preference. The groups were designated: Group A (high compliance, long slow distance), Group B (sports), and Group C (low compliance, active control). Group A was composed of fourteen subjects, while Groups B and C consisted of six subjects each. Initial screening data was incorporated into the pre-training data. A personal file was established for each subject.

Initial Screening

Initial screening tests were conducted in the Human Performance Laboratory at the University of Montana beginning in mid November 1978, and were concluded by the quarters end. Subjects were scheduled to report to the laboratory by appointment. The subject was asked to peruse various printed materials, including a brief outline of the lifestyle program and its requirements. Initial questions were answered and the subject was asked to sign an informed consent form (appendix). The following initial screening tests were then conducted:

1. Resting blood pressure
2. Height and weight
3. Tricep, chest, and abdominal skinfolds were measured on male subjects, and the tricep and illiac crest were measured on female subjects. Measurements were made using a Lange skinfold caliper, and were repeated in an attempt to obtain the highest degree of accuracy. Percentage body fat was estimated from these values using the nomogram of Consolazio, (appendix).
4. Measurements of the flexed bicep were recorded in centimeters.

Subjects concluded the initial screening after completing a University of Montana health risk analysis, and a personal lifestyle questionnaire.

Subjects were then given appointments to report to the University of Montana Student Health Service, for the drawing of a blood sample. Appointment forms, emphasizing the need for a twelve hour fast prior to the collection of the blood sample, were presented to each subject.

Blood samples were collected between the hours of 8:00 and 9:00 A.M. All blood collection was conducted by the student health service staff, under the supervision of Dr. John Bruckner, the physician associated with the project. Blood samples were then forwarded to the Metpath Corporation, Portland, Oregon division, for analysis. This analysis resulted in a blood profile for each subject.

Appointments were then scheduled for fitness testing to be conducted at the Human Performance Laboratory, University of Montana. Maximum oxygen uptake was measured directly by a Technology Incorporated oxygen consumption computer. The test was conducted on a Quinton model 18-60 treadmill accompanied by a Quinton model 643 automatic program control. The exercise electrocardiogram was monitored by an Avionics stress test monitor.

- a. The subject was instructed as to the testing procedure.
- b. Disposable ECG electrodes were affixed in a modified V-5 configuration.
- c. The subject engaged in a five minute warmup, to familiarize him with the treadmill and to prepare him for the program.
- d. The subject was fitted with headgear, including nose clip and one-way breathing valve.
- e. A modified Balke protocol was employed. Speed of the treadmill was set at 3.4 miles per hour. The treadmill was set at 4% grade in the initial stage

of the program. The program consisted of ten, two minute stages, with the grade increasing in increments of two percent with each stage.

- f. The oxygen consumption computer was engaged during the second minute of the fifth stage for males, and during the second minute of the third stage for females. The computer was then engaged whenever the test administrator deemed necessary. This depended on the condition of the individual subject. It was our intention to obtain at least two sub-maximal readings. The subject was instructed to let the administrator know, through the use of a hand signal, when he had one minute of exercise left. The computer was then engaged during the final minute of exercise.
- g. The grade of the treadmill was gradually reduced using the manual control, and the subject was allowed to cool down, as his heart rate and blood pressure were monitored.
- h. Heart rate was continuously monitored during the program, and was recorded at the conclusion of each stage. Blood pressure was checked and recorded at various intervals throughout the procedure.

Dr. John Bruckner was present during the testing of all subjects age thirty-five or above. A twelve lead resting electrocardiogram was administered and evaluated by Dr. Bruckner prior to the fitness testing of these subjects.

Upon completion of the fitness test, each subject completed an Interhealth health risk analysis questionnaire. The completed forms were then forwarded to the Interhealth Corporation, San Diego, California. This yielded a computerized readout for each subject. The readout identified, and listed in order of precedence, the health risks of that particular subject, and offered suggestions for the reduction of those risks.

TABLE 1

PHYSICAL CHARACTERISTICS OF SUBJECTS

Subject	Age (yrs.)	HT (CM)	WT (KG)
ED	47	176.0	101.8
RS	23	190.5	117.3
JT	40	174.0	93.0
JB	33	178.0	83.2
HJ	28	190.0	81.3
JD	42	173.0	79.5
JDO	42	175.0	90.5
DW	43	178.0	97.0
CM	27	178.0	74.5
DM	41	180.0	79.0
TV	30	193.0	117.0
JDR	39	172.0	71.4
GK	25	177.0	95.0
TH	39	184.0	91.8
SR	42	176.0	86.3
AB	38	180.0	89.5
PG	26	176.0	62.7
ML	33	162.0	60.9
KP	26	163.0	58.6
SM	30	164.0	64.0
CH	38	160.0	55.0
JDOL	42	180.0	92.7
SS	42	178.0	76.0
BB	36	183.0	70.0
TS	35	178.0	90.0
FS	44	176.0	81.0
MEANS: 26	35.8	176.0	83.2

TRAINING

Training began on January 8, 1979 and lasted twelve weeks. The training program consisted of three supervised training sessions per week. Sessions were held on Monday, Wednesday and Friday at 12:00 noon, and were one hour in duration. Subjects were instructed to meet in the Field House annex, University of Montana, at the specified time. Basic flexibility exercises were demonstrated and introduced to each subject individually, prior to beginning the jogging program. The jogging program was initiated with either a one mile run or a one mile run/walk. Correct running techniques were introduced and each subject's gait was evaluated, with necessary corrections being made. Each subject was asked to keep a written record of the training period, on a personal performance/workout log (appendix).

Group A (high compliance, long slow distance) was composed of fourteen subjects who regularly attended the training sessions. Long duration, low intensity running was emphasized throughout the training period. Subjects initiated the training with a one mile run. At the conclusion of the twelve week training period all subjects in Group A were running between four and six miles, three times per week. Intensity was estimated to be between an eight and ten minute mile pace.

Group B (sports) consisted of six subjects who chose to participate in racquetball and basketball. The racquetball and basketball, a combination of high and low intensity, varied with the skill level of the subjects. Three of the subjects engaged in half court basketball and racquetball, while the remaining three subjects devoted their time to

full court basketball.

Group C (low compliance, active control) consisted of six subjects who, after participating during the pre-training period, did not comply with the entire training program. Because some of the subjects participated during the initial weeks of training, or engaged sporadically in training, this group was designated active control, and was established to provide a means of comparing the effects of the training program.

Post Testing

Within two weeks of the completion of the twelve week training program all subjects reported to the Human Performance Lab for post testing. The same tests were conducted in the same manner as described under initial screening. The subjects were also scheduled to report to the student health service for the drawing of a blood sample.

Chapter 4

ANALYSIS AND DISCUSSION OF RESULTS

This chapter presents the data obtained, statistical analysis of the data, and a discussion of the results and their relationship to other studies reported in the literature.

COMPARISON OF CHANGES WITHIN TRAINING GROUPS

A one-way analysis of variance was employed to compare pre and post training values between the three Groups: A, B, and C. Pearson Product Moment Correlation Coefficients were computed between parameters of interest. The significance level was set at .05 for all statistical treatments.

Table 2 presents the means, standard deviations, pre to post change, and percent change for all subjects. Tables 3, 4, and 5 present the same information for Groups A, B, and C. The results of these analyses follow.

HDL CHOLESTEROL

The mean pre to post training difference for all subjects was 3.58 mg/dl. This difference was not statistically significant. The mean difference in HDL for Group A was 11.4 mg/dl, for Group B -6.7 mg/dl, and for Group C -4.7 mg/dl. Analysis of variance revealed no significant differences between the pre-HDL values of Groups A, B, and C,

however the difference between Groups A and B (9.8 mg/dl) approached significance with an F value of 2.1.

A highly significant difference was observed reflecting change in HDL between Group A and Groups B and C. The calculated F value of 19.3 falls beyond the .001 level of significance.

Group A experienced a substantially greater percent increase in HDL, 22.4 percent versus -11.0 and -7.9 percent for Groups B and C respectively. This result may be expected due to the lower pre-training value (50.8 mg/dl) of Group A compared to the pre-training values of Group B (60.6 mg/dl) and Group C (59.0 mg/dl). A correlation coefficient of $-.50$, significant beyond the .01 level, was found between the pre training level of HDL, and the change in HDL.

Among the total of twenty-six training subjects, twelve decreased, and fourteen increased in HDL levels. The largest increases, 29.0 mg/dl or 57.5 percent, 19.0 mg/dl or 36.5 percent, and 19.0 mg/dl or 57.5 percent were observed in subjects K.P., S.M., and J.T. respectively. Tables containing pre and post training data for individual subjects may be found in the appendix.

All subjects whose increases are reported above, began with a pre HDL level close to or below the mean pre HDL level of Group A (50.8 mg/dl). In lieu of this observation it may be of interest to examine changes in the HDL level of subject M.L., also a member of Group A. Subject M.L. experienced an increase of 15 mg/dl or 21.4 percent, but her initial level of HDL was 70 mg/dl, which is far in excess of the group mean, and may be associated with a high degree of cardioprotection,

TABLE 2

EFFECTS OF TRAINING ON ALL GROUPS

(A+B+C) N=26

	Pre mean (SD)	Post mean (SD)	Change	%Change
HDL Cholesterol (mg/dl)	55.0 (19.30)	58.5 (10.63)	3.5 (11.01)	6.36
Total Cholesterol (mg/dl)	214.8 (46.96)	227.0 (55.10)	12.2 (35.28)	5.67
Oxygen Uptake (ml/kg/min)	39.3 (7.39)	46.6 (9.88)	7.3 (7.18)	18.57
Weight (kg)	83.2 (16.10)	81.4 (16.03)	-1.8 (1.57)	-2.16
Percent Body Fat	19.1 (6.65)	16.4 (5.39)	-2.7 (3.28)	-14.13
Triglycerides	137.0 (62.06)	142.0 (59.61)	4.9 (46.29)	3.57
HDL Cholesterol/ Total Cholesterol Ratio	0.265 (.071)	0.270 (.092)	.004 (.051)	1.50

TABLE 3
EFFECTS OF TRAINING ON GROUP A
(HIGH COMPLIANCE, LONG SLOW DISTANCE)

N=14

	Pre mean (SD)	Post mean (SD)	Change	%Change
HDL Cholesterol (mg/dl)	50.8 (10.29)	62.3 (12.28)	11.4 (8.64)	22.44
Total Cholesterol (mg/dl)	197.8 (47.32)	223.7 (59.54)	25.9 (33.88)	13.09
Oxygen Uptake (ml/kg/min)	40.5 (6.55)	48.2 (4.04)	7.7 (6.48)	19.01
Weight (kg)	74.4 (12.85)	72.7 (12.07)	-1.7 (1.54)	-2.28
Percent Body Fat	19.6 (18.16)	17.2 (6.10)	-2.4 (2.92)	-12.24
Triglycerides	135.9 (67.81)	142.9 (73.30)	7.0 (45.66)	5.15
HDL Cholesterol/ Total Cholesterol Ratio	0.269 (.084)	0.299 (.102)	.030 (.046)	11.15

TABLE 4

EFFECTS OF TRAINING ON GROUP B
(SPORTS: BASKETBALL, RACQUETBALL)

N=6

	Pre mean (SD)	Post mean (SD)	Change	%Change
HDL Cholesterol (mg/dl)	60.6 (10.19)	54.0 (8.05)	-6.7 (3.72)	-11.05
Total Cholesterol (mg/dl)	219.0 (36.43)	208.2 (42.75)	-10.8 (14.30)	-4.93
Oxygen Uptake (ml/kg/min)	42.1 (10.12)	50.9 (16.89)	8.8 (9.57)	20.9
Weight (kg)	91.5 (15.04)	89.0 (15.21)	-2.5 (1.04)	-2.73
Percent Body Fat	15.3 (3.32)	14.0 (5.51)	-1.3 (3.61)	-8.49
Triglycerides	117.3 (48.87)	118.3 (43.14)	1.0 (19.11)	.852
HDL Cholesterol/ Total Cholesterol Ratio	0.284 (.069)	0.251 (.088)	-.033 (.043)	-11.61

TABLE 5

EFFECTS OF TRAINING ON GROUP C
(LOW COMPLIANCE, ACTIVE CONTROL)

N=6

	Pre mean (SD)	Post mean (SD)	Change	%Change
HDL Cholesterol (mg/dl)	59.0 (6.78)	54.3 (4.80)	-4.7 (4.27)	- 7.96
Total Cholesterol (mg/dl)	250.3 (30.08)	253.8 (53.05)	3.5 (42.18)	1.39
Oxygen Uptake (ml/kg/min)	33.9 (3.30)	38.5 (7.31)	4.7 (6.71)	13.86
Weight (kg)	95.5 (12.66)	94.1 (14.07)	-1.3 (2.06)	- 1.36
Percent Body Fat	21.8 (3.18)	17.0 (2.96)	-4.8 (3.25)	22.01
Triglycerides	159.8 (61.82)	163.8 (26.31)	4.0 (69.97)	2.50
HDL Cholesterol/ Total Cholesterol Ratio	0.238 (.033)	0.221 (.044)	-.018 (.041)	- 7.56

according to data compiled at Framingham (25).

Although the change in HDL level for the total sample was not statistically significant, the trend towards increasing levels following training is consistent with results reported by Vial (47), Webster (50), Wood (54), and Washburn (49).

The highly significant difference in HDL change between Group A, and Groups B and C is worthy of close scrutiny. According to data compiled by Castelli (10), the increase of 11.4 mg/dl or 22.4 percent observed in Group A, is associated with a approximately 40% reduction in coronary heart disease risk, over the twelve week training period.

Correlation coefficients were computed between parameters of interest within Group A. A significant negative correlation ($r = -.52$) existed between the change in HDL and the change in weight. A positive correlation, between change in HDL and change in maximum oxygen uptake ($r = .35$) approached significance at the .10 level.

Further analysis of all groups revealed no significant correlations between either pre, post or change in HDL, and any other parameters of interest.

TOTAL CHOLESTEROL

The mean change in total cholesterol, observed among all training subjects was 12.2 mg/dl.

Groups A and C experienced increases in total cholesterol of 25.9 mg/dl and 3.5 mg/dl respectively. Group B experienced a decrease of 10.8 mg/dl.

Ten of the training subjects decreased, fifteen increased, and one showed no change in total cholesterol. Subjects T.S. and S.S. showed the largest increases, 91 mg/dl and 86 mg/dl respectively, and subjects A.B. and E.D. recorded the largest decreases, 24 mg/dl and 21 mg/dl respectively. Subject J.D. had the highest pre training value of 305 mg/dl and the highest post training value of 366 mg/dl. No significant correlation was found between the total cholesterol level and percent body fat.

Analysis of all groups, revealed no significant correlations between total cholesterol and other parameters of interest. Further examination did reveal that positive correlations between total cholesterol change and triglyceride change ($r = +.29$), and total cholesterol change and HDL cholesterol change ($r = +.29$) approached significance.

The increase in total cholesterol of 25.9 mg/dl or 13.0 percent observed in Group A is contrary to the substantial amount of literature reporting increased levels of HDL with simultaneous decreases, or no mean changes, in the total cholesterol measurement following training. This observation therefore requires further analysis.

The subjects in Group A who contributed most to the 13.0 percent increase in total cholesterol were subjects T.S., S.S., K.P., D.M., and J.Dol. The pre to post increases in total cholesterol of these subjects were 91 mg/dl, 86 mg/dl, 42 mg/dl, 41 mg/dl and 37

mg/dl respectively. The mean difference in total cholesterol in Group A, excluding these five subjects, is 7.0 mg/dl. Subjects K.P., J.Dol, and D.M. consequently showed increases in HDL of 53.7, 57.5 and 45.4 percent respectively, however subjects T.S. and S.S. recorded increases in HDL of only 4.0 and 2.3 percent.

Since the total cholesterol measurement is the sum of the cholesterol contained in VLDL, LDL, HDL, and chylomicrons, a simultaneous decrease in total cholesterol with increasing HDL must be accompanied by decreases in VLDL and LDL cholesterol. Results consistent with this hypothesis have been reported by Lopez (31) and Carlson and Mossfeldt (9). Very low density lipoproteins are glyceride-rich lipoproteins that carry endogenous triglycerides, and low density lipoproteins are lipoprotein remnants partially or completely resulting from metabolism of VLDL. Both are acutely affected by diet, and more specifically by the intake of saturated fat. Therefore an increase of saturated fat in the diet, and drastic increases in the HDL fraction, as observed in subjects K.P., J.Dol, and D.M., should be reflected in an elevated total cholesterol concentration.

The diet of training subjects was not controlled. It is possible that elevated total cholesterol levels reflected a change in diet, and/or an increase in saturated fat. This is proposed as one possible explanation, since no complete record of the caloric intake of subjects existed.

In light of the increase in total cholesterol of 91 mg/dl, and the post total cholesterol value of 366 mg/dl of subject T.S., the

possibility exists that some subjects failed to observe the twelve hour fast as instructed. There is also the possibility of discrepancy in the pre to post determination of the total cholesterol level. All laboratory determinations were carried out by the Metpath Corporation, hence the author had no control over laboratory processes.

Because of scheduling problems precipitated by the spring break, approximately eight post training blood samples were drawn two weeks after the conclusion of the training period. All eight of these subjects increased in total cholesterol. Rochelle (39) reported that plasma cholesterol levels returned to pre training levels within four weeks after the cessation of training. This delay in the collection of the post blood sample coupled with a possible change in diet, may explain the increased total cholesterol levels of the above mentioned subjects.

HDL CHOLESTEROL/TOTAL CHOLESTEROL RATIO

Data gathered from recent epidemiological studies (10,24,) has indicated that the HDL/total cholesterol ratio may perhaps be a more accurate predictor of inverse cardiovascular risk than the lone HDL determination. In response to these findings the HDL/total cholesterol ratio was calculated for all training subjects.

The mean difference pre to post training for all subjects was .004 mg/dl. The mean difference in HDL/total cholesterol ratio for Group A was .030 mg/dl, for Group B -.033 mg/dl, and Group C -.018 mg/dl.

Group A experienced an 11.2 percent increase in HDL/total

cholesterol ratio, versus -11.5 and -7.5 percent for Groups B and C respectively. Among the total of twenty-six training subjects, ten decreased, and sixteen increased their HDL/total cholesterol ratios. The greatest increase, .114 mg/dl, was observed in subject T.H., while the largest decrease, .100 mg/dl, was found in subject J.T.

A significant positive correlation ($r = +.44$) between change in HDL/total cholesterol ratio and the change in maximum oxygen uptake, was also uncovered while gleaning Group A's data.

Although Group A showed a 13.0 percent increase in total cholesterol, an increase of 22.5 percent in HDL resulted in an increase of 11.2 percent in the HDL/total cholesterol ratio. If, as eluded to earlier, subjects in Group A increased their intake of saturated fats, increases in HDL and in the HDL/total cholesterol ratio may have better equipped them to manage the elevated level of total cholesterol. In contrast, Group B, which recorded a 4.9 percent decrease in total cholesterol, and Group C, which showed a slight increase (1.3 percent), both registered decreases in both HDL and the HDL/total cholesterol ratio.

OXYGEN UPTAKE

There was an increase in mean maximum oxygen uptake of 7.3 ml/kg/min among all exercising subjects. Group A showed an increase of 7.7 ml/kg/min or 19.0 percent. Group B recorded the largest increase of 8.8 ml/kg/min, which translates to a 20.9 percent change. Group C increased 4.7 mg/kg/min, or 13.8 percent.

Pre training values in Groups A, B and C were 40.5, 42.1 and

33.9 respectively. A majority of the literature has reported negative correlations between the pre training level of maximum oxygen uptake and the change in maximum oxygen uptake. Group C, whose pre training value (33.9 ml/kg/min) was lowest among the groups, recorded the smallest increase (4.7 ml/kg/min). However it must be noted that this group was designated active control and thus the sporadic physical activity of this group did not match that of the training groups. Analysis of variance revealed no significant differences in the pre maximum oxygen uptake values between Groups A, B, or C. The difference between Groups A and C (7 ml/kg/min) approached significance with an F value of 2.5.

A significant difference ($F = 3.2$) was noted between Group A and Group C in terms of post maximum oxygen uptake; however no significant difference was found in maximum oxygen uptake change within Groups A, B, and C.

Analysis within training Group A, revealed a significant positive correlation ($r = .44$) between the change in maximum oxygen uptake and the change in the HDL/total cholesterol ratio. There were no other significant correlations between the change in maximum oxygen uptake and any other parameter of interest.

CALORIC EXPENDITURE

Examination of individual workout logs, enabled us to calculate the number of calories per week of vigorous activity (> 7.5 cal/min) expended by each training subject. Although subjects were asked to be as accurate as possible in the recording of physical activity,

entries in the log were subjective. An evaluation of the workout logs left the author skeptical of the accuracy of some of the recordings, however he did conclude that the majority of the recordings were indeed accurate.

The following chart lists the mean weekly caloric expenditure, the initial maximum oxygen uptake values, and the change in maximum oxygen uptake for each training group.

	mean weekly caloric expenditure vigorous activity (> 7.5 cal/min)	initial maximum oxygen uptake (ml/kg/min)	change in maximum oxygen uptake (ml/kg/min)
Group A	1165	40.5	7.7
Group B	1416	42.1	8.8
Group C	933	33.9	4.7

Highly significant positive correlations existed between pre maximum oxygen uptake and calories per week ($r = .39$), between post maximum oxygen uptake and calories per week ($r = .74$), and between the change in maximum oxygen uptake and calories per week ($r = .62$). Although the above data is logical, possible explanations for the differences in changes in maximum oxygen uptake noted between the three training groups must be further discussed.

Thirteen subjects in Group A increased in maximum oxygen uptake, and one subject recorded no change. Group A expended 1165 calories per week of vigorous activity. This expenditure was associated with an increase of 19.0 percent in maximum oxygen uptake, and a 22.4 percent increase in high density lipoprotein cholesterol. Data

indicates that the long duration, low intensity exercise performed by Group A, over the twelve week training period, was of sufficient duration and intensity to elicit a training effect. This may partially be due to the relatively low initial levels of maximum oxygen uptake observed in this group.

Group B, expended 1416 calories per week of vigorous activity and exhibited the largest increase (8.8 ml/kg/min), in maximum oxygen uptake of the three groups. Subjects H.J., C.M., and G.K., significantly increased their maximum oxygen uptakes, while the remaining three subjects essentially remained the same. Examination of the training logs indicates that those subjects who significantly increased in maximum oxygen uptake either participated in full court basketball, or were competitive racquetball players whose skill level enabled them to play with a greater intensity. The weekly caloric expenditure of these subjects account for the mean value of 1416 calories per week reported by Group B subjects. The subjects whose maximum oxygen uptake remained the same either engaged in half court basketball, or were new to the sport of racquetball.

It appears that the workloads encountered during full court basketball and competitive racquetball, played over the twelve week training period, were of sufficient duration and intensity to elicit a training effect. The more sedentary half court basketball and beginning racquetball lacked the intensity required to produce such an effect.

Group C expended 933 calories per week in vigorous activity

and showed a mean increase of 4.7 ml/kg/min in maximum oxygen uptake. Three subjects in this group increased, two subjects recorded no change, and one subject decreased in maximum oxygen uptake over the twelve week period.

It may have been possible that the sporadic physical activity engaged in by some members of this active control group, may have been enough to raise the very low initial level of maximum oxygen uptake. However the low initial mean level of maximum oxygen uptake, coupled with some highly significant increases in this value, especially in subject A.B., caused us to wonder if we actually obtained true maximum samples during our pre test procedures. In any stress testing situation, unfamiliarity of subjects with testing, and anxiety due to the testing environment, may hamper the efforts of an experimenter attempting to obtain a true maximum.

PERCENT BODY FAT

There was an overall reduction in percent body fat of 2.7 among all exercising subjects. Reductions of 2.4, 1.3, and 4.8 occurred in Groups A, B, and C respectively. Analysis of variance revealed no significant differences in pre or post percent body fat between Groups A, B, and C. There were no significant differences between Groups A, B, and C in percent body fat change, however the difference between Groups B and C (6.5) approached significance ($F = 2.0$).

BODY WEIGHT

There was a mean reduction in body weight of 1.8 kilograms among all subjects. Reductions of 1.7, 2.5, and 1.3 kilograms were recorded in Groups A, B, and C respectively. Analysis of variance revealed significant differences in pre body weight ($F = 6.8$), and post body weight ($F = 6.7$) between Group A and Groups B and C. However there was no significant difference in body weight change within the three groups.

A significant negative correlation ($r = -.46$) existed between maximum oxygen uptake and body weight. Further analysis revealed a significant negative correlation between weekly caloric expenditure (vigorous exercise) and change in body weight ($r = -.33$). A highly significant positive correlation existed between the change in body weight and the change in triglycerides ($r = .51$).

TRIGLYCERIDES

A mean increase in triglycerides of 4.9 was recorded among all exercising subjects. Mean increases of 7.0, 1.0, and 4.0 occurred in Groups A, B, and C respectively. There were no significant differences in either pre or post triglyceride levels, between Groups A, B, and C. No significant differences in triglyceride change existed within the three groups. Analysis revealed a significant negative correlation ($r = -.39$), between the triglyceride level and maximum oxygen uptake.

DISCUSSION

Four of the twenty-six subjects were female, all of whom were members of Group A (high compliance, long slow distance). The women were relatively sedentary prior to the training program. The female pre training mean for maximum oxygen uptake was 36.0 ml/kg/min, slightly less than the overall pre training mean of approximately 40 ml/kg/min. As members of Group A, these women enthusiastically adhered to the requirements of the training program. The mean change in maximum oxygen uptake of 14.0 ml/kg/min, recorded among female subjects, was twice as great as the overall change of 7.3 ml/kg/min. While the body weight of female subjects remained essentially the same, the reduction in percent body fat experienced by the women was again twice that of the overall change. This may be explained by the relatively high female pre fat mean of 31.0 percent.

The mean change in HDL of 17.0 mg/dl, recorded by female subjects, was higher than the overall mean change of 3.5 mg/dl. A mean increase of .058 mg/dl in the HDL/total cholesterol ratio was also recorded among the women, compared to the overall increase in the ratio of .004 mg/dl.

The hypothesis that elevated plasma concentrations of HDL exert a protective effect against coronary heart disease (CHD) is in accord with the relative immunity to CHD of pre menopausal women, whose HDL concentrations are about thirty to sixty percent higher than those of male counterparts in the same age groups. The fact that women have higher HDL levels than men, may explain why women have lower rates of CHD.

Our data indicates a possible difference in response to training between male and female subjects, reflected by the change in HDL and the HDL/total cholesterol ratio. Recent studies have substantiated the original work of Barr (2), which showed that estrogens raise levels of HDL. Perhaps it was the presence and/or level of estrogen which accounted for the difference in response to training.

Female subjects in Group A ran as far and almost as fast as their male counterparts. Since the female initial fitness level was lower, their training was relatively more intense. Sharkey (41) has shown that increases in maximum oxygen uptake are inversely related to initial level of fitness ($r = -.54$), and this study found the same to be true of HDL. Thus the greater changes in maximum oxygen uptake exhibited by the female subjects may merely be a reflection of their previous inactivity.

A sparse amount of literature is available pertaining to the effects of different types of exercise on HDL. Indications point to the duration of the exercise as the key factor initiating significant changes. In this instance duration is perceived not only in terms of the length of each exercise session, and number of kilo calories expended, but also the period of time over which the activity has been undertaken. Studies that have shown exercise to be associated with higher levels of HDL (9, 50, 54, 47) have studied exercise that was not only of the long duration type, but which had been carried out over an extended period of time.

Wood (54) reported higher HDL and HDL/LDL ratios in forty-one

male distance runners who averaged at least fifteen miles per week during the previous year, and were in active training.

Webster (50) studied thirty middle aged men who participated in forty-five minutes of jogging, three days per week for twelve weeks. He too found the joggers to have the higher levels of HDL than the controls.

Vial (47) conducted a seven week exercise program consisting of thirty minutes of aerobic activity per day. Serum electrophoresis revealed a decrease in beta and pre beta lipoproteins, with an increase in the alpha lipoprotein fraction.

In this study it was hypothesized that exercise Group A (high compliance, long slow distance), due to the low intensity, long duration exercise, would be more likely to experience beneficial changes in HDL, then would Groups B (sports), and C (low compliance, active control). This was in fact the case.

Three investigations dealing with the effects of exercise and other parameters on the high density lipoprotein fraction have been conducted at the University of Montana during the past four years.

Washburn (49) investigated the effects of intensity and duration of training on HDL. Exercise prescriptions were individualized for each subject, and all training was conducted on a treadmill. Two training groups were identified: Group A (low intensity, long duration), and Group B (high intensity, short duration). No significant change in HDL within either training group was reported. The initial level of HDL was significantly correlated with the change in HDL.

Several factors, additively, may have accounted for the lack of significant change. Each training group was composed of five fit, young, undergraduate students. The subjects trained three days per week for seven weeks.

In this most recent investigation we were careful to select middle-aged, relatively sedentary subjects, and the training period was lengthened to twelve weeks. These changes may have precipitated the significant changes in HDL observed in this study.

Simpson (42) conducted an association study, in which he attempted to identify factors associated with the high density lipoprotein fraction. The major finding in this study was that HDL was found to be highly related to vigorous physical activity ($r = .58$), but not to total activity. These results were similar to those reported by Paffenbarger (35), who showed that subjects who expended less than 2000 calories per week in vigorous activity (> 7.5 cal/min) were 64% more likely to suffer heart attacks.

The present investigation revealed a very high positive correlation between the change in maximum oxygen uptake and the weekly caloric expenditure consisting of vigorous activity ($r = .62$).

Although no significant correlations between change in HDL and weekly caloric expenditure, (vigorous activity > 7.5 cal/min), existed in this study, a mean caloric expenditure of 1165, was associated with an increase of 19.0 percent in maximum oxygen uptake, and an increase of 22.4 percent in high density lipoprotein cholesterol.

All three training groups experienced mean increases in maximum

oxygen uptake. Our data indicates that the long duration, low intensity exercise engaged in by Group A, and the full court basketball and competitive racquetball performed by three members of Group B was of sufficient duration and intensity to elicit a training effect.

As discussed earlier, the mean change in total cholesterol of 12.2 mg/dl, and more specifically the increase of 25.9 mg/dl observed in Group A, is contrary to the majority of literature reporting increased levels of HDL with simultaneous decreases in total cholesterol following training. In addition to the increase in total cholesterol observed in Group A, an increase of 22.4 percent in HDL resulted in an increase of 11.2 percent in the HDL/total cholesterol ratio.

In light of the evidence of the cardio protective factor associated with higher levels of HDL (1, 10, 34, 38) the role of exercise in providing cardio protection via its action on serum cholesterol needs re-evaluation. If observations made in this and other studies hold true, it is possible that exercise may be effective in reducing the risk of coronary heart disease without lowering total serum cholesterol levels, especially if these changes can be elicited by low intensity activity undertaken over an extended period of time. It is possible that an increased level of HDL is an important factor in the low incidence of coronary heart disease among those engaged in extensive amounts of activity either leisure time or occupational.

Additional investigation must be done before it can be stated without doubt, that low intensity, long duration exercise is the most efficient means of increasing levels of high density lipoprotein. Such

a finding, however, would be a godsend to those in fitness related fields. Exercise of low intensity and gradually increasing duration could be engaged in with a minimum of discomfort, and therefore might not discourage an individual beginning a fitness program after years of sedentary living. Low intensity, long duration exercise could form the foundation on which a lifelong program of exercise could be built.

Chapter 5

SUMMARY, CONCLUSIONS, RECOMMENDATIONS

SUMMARY

This study investigated the effects of duration of training on the levels of HDL and related serum lipids, in a sample of middle aged, relatively sedentary men and women.

Twenty-six subjects who met the criteria for participation in this study were placed into two training groups, according to their personal preference. The groups were designated: Group A (high compliance, long slow distance), and Group B (sports). A third group (C) was composed of subjects who did not fully comply with the training program (low compliance, active control). Subjects in the training groups engaged in twelve weeks of either low intensity, long duration jogging, or basketball and racquetball. Exercise sessions were one hour in duration and were scheduled three days per week. Following the twelve week training period the pre and post training measurements were compared within each training group and between training groups.

The post training mean difference in HDL for all subjects was not statistically significant. A highly significant difference in HDL change, existed between Group A and Groups B and C. The substantial increase in Group A, is associated with a 40% reduction in coronary heart disease risk. The initial level of HDL was significantly correlated with change in HDL.

Groups A and C experienced increases in total cholesterol, while Group B showed a decrease in the total cholesterol level. A slight increase in the HDL/total cholesterol ratio was observed among the combined training groups. Group A experienced a substantially greater percent increase in the HDL/total cholesterol ratio than did Groups B and C.

There was a significant increase in mean maximum oxygen uptake among the combined training groups. All groups showed increases in maximum oxygen uptake, with Group B experiencing a slightly greater increase than Group A. Significant correlations existed between post maximum oxygen uptake and pre cholesterol and between pre maximum oxygen uptake and pre percent body fat. A highly significant correlation was observed between the change in maximum oxygen uptake and the weekly caloric expenditure (vigorous activity, >7.5 cal/min).

There was an overall reduction in body weight and percent body fat among all exercising subjects, however these changes were not statistically significant. A mean increase in triglyceride level was detected among the combined training groups.

CONCLUSIONS

The results of this study support the following conclusions:

- A. Low intensity, long duration exercise had a significant effect on the HDL levels of those relatively sedentary, middle aged subjects who complied with a twelve week training program.

- B. The initial level of HDL is inversely related to the amount of change that can be expected as a result of exercise training.
- C. A twelve week program of low intensity, long duration exercise, full court basketball and competitive racquetball is of sufficient duration and intensity to elicit a training effect on relatively sedentary, middle-aged subjects.
- D. The change in maximum oxygen uptake is related to the weekly caloric expenditure derived from vigorous physical activity (> 7.5 cal/min).

RECOMMENDATIONS

Based on the results of this study, the following recommendations for further study are proposed:

- A. Controlled training studies, composed of subjects with low initial levels of HDL and maximum oxygen uptake, should be conducted over longer periods of time utilizing measures of all the lipoprotein classes. In studies of this type caloric intake and diet should be controlled.
- B. The HDL levels of individuals who have engaged in different types of physical activity over extended periods of time should be compared.

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APPENDIX A

INFORMED CONSENT FORM

PROJECT LIFE/STYLE

Consent Form

This program will help you examine your lifestyle, identify your major health risks and take positive steps for improvement.

The program includes:

- a health risk analysis
- a blood profile
- personal consultation
- fitness tests
- fitness program
- follow up tests

As a participant you will receive valuable insights concerning your health and lifestyle, professional medical, exercise and diet advice, improved fitness, and a reduction in your 10 year risks of death.

In return we ask that you make a personal commitment to full participation, including tests and training sessions. You should continue your training when meetings or travel make it impossible to attend regular training sessions and attend make-up sessions when necessary.

Project data will be coded to insure confidentiality. No data will be released without your consent (e.g. to your personal physician).

We look forward to your participation and remain ready to answer your questions concerning any aspect of the project.

I have read the preceeding explanation and consent to full participation in the program.

Signature

Date

APPENDIX C

MODIFIED BALKE TREADMILL PROTOCOL

STAGE	SPEED (miles per hour)	DURATION (Minutes)	PERCENT GRADE
1	3.4	2	4
2	3.4	2	6
3	3.4	2	8
4	3.4	2	10
5	3.4	2	12
6	3.4	2	14
7	3.4	2	16
8	3.4	2	18
9	3.4	2	20
10	3.4	2	22

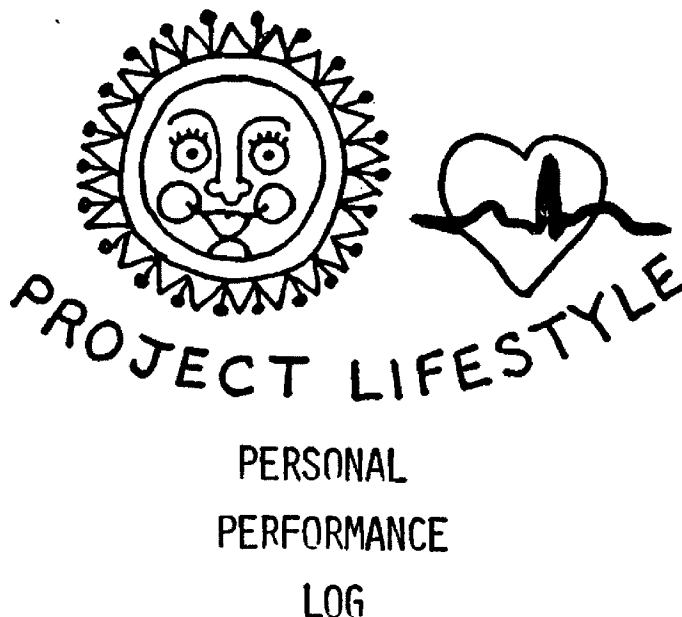
APPENDIX D

PERSONAL PERFORMANCE LOG

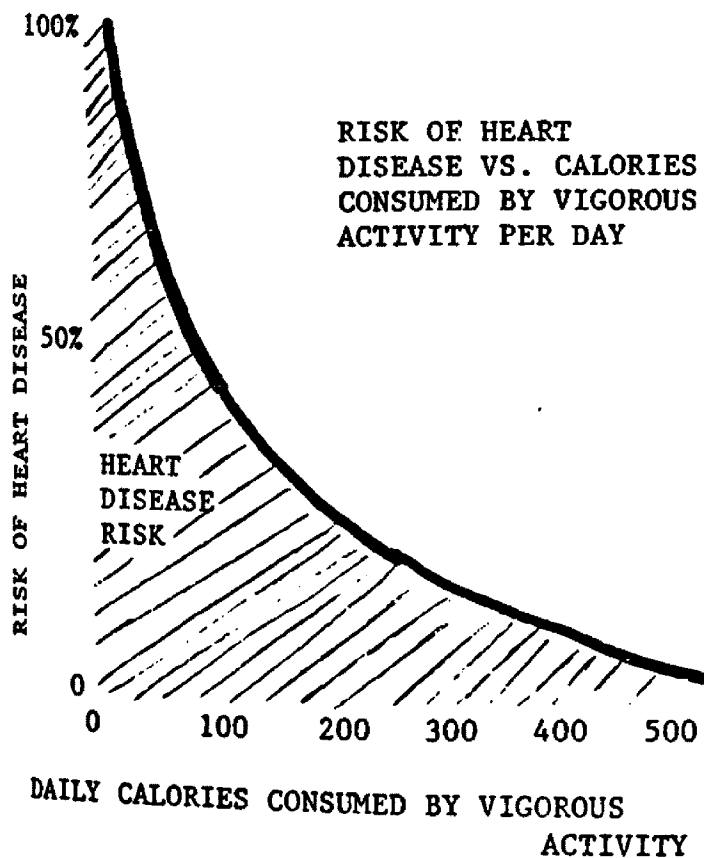
HEALTH

Health and longevity are associated with these health habits:

- * Adequate sleep (7-8 hours per night)
- * Good breakfast
- * Regular meals (3 or more a day) Avoid snacks
- * Maintain desirable weight
- * NO smoking
- * Drink moderately (1 to 2 drinks a day) or not at all
- * Regular physical activity



YOUR LIFESTYLE HAS A GREAT DEAL MORE TO DO WITH WHAT MAKES YOU SICK AND WHEN YOU DIE THAN ALL THE INFLUENCES OF MEDICINE.



APPENDIX E

PROCEDURE FOR TOTAL CHOLESTEROL DETERMINATIONS

Laboratory total cholesterol determinations were performed by MetPath Inc., Portland Oregon, Service Center.

The Hycel method for the determination of total cholesterol makes use of the Lieberman-Buchard reaction. The addition of concentrated sulfuric acid to a mixture of cholesterol and acetic acid produces a green color. The Hycel Stable Cholesterol Reagent, combines the sulfuric acid and acetic acid with acetic anhydride to form a single reagent.

The Hycel cholesterol test is calibrated with Hycel Cholesterol Standard, a primary standard containing 200 mg/dl of cholesterol in glacial acetic acid. The test requires only one calibration point. Hycel Normal Lipid Reference serum is used as a control.

Specimen Requirement:

1 ml. serum, fasting specimen is recommended

Test no. 025

APPENDIX F

PROCEDURE FOR SEPARATION OF HIGH DENSITY

LIPOPROTEIN FROM SERUM

Laboratory high density lipoprotein cholesterol determinations were performed by MetPath Inc., Portland Oregon, Service Center.

At MetPath, HDL is separated from other lipoproteins by precipitation with a standardized solution of heparin-manganese under controlled conditions. The cholesterol present in the fraction associated with HDL is then quantified. HDL determinations are rigidly controlled. A quality control serum in the low, middle, and high HDL range is analyzed in the beginning and at the end of each batch of twenty-five specimens. The accuracy of the MetPath test has been established by the use of a serum pool obtained from the Lipid Standardization Laboratory of CD.

Specimen Requirement:

3 ml serum or 3 ml plasma (EDTA). A fasting specimen is recommended.

Test No. 204.

CORONARY HEART DISEASE RISK

How to predict the risk of CHD

The tables below show the probable risk of developing coronary heart disease associated with various approximate total-cholesterol and HDL-cholesterol levels. In both tables, the risk is compared to a standard with an index value of 1.00.

To determine the risk of CHD in a middle-aged patient, simply multiply the risk index for his or her total cholesterol by the index for HDL cholesterol. Thus, a man with a total cholesterol of 300 mg% and an HDL cholesterol of 25 mg% has a risk index of 3.00×2.00 , or six times the standard risk of developing CHD. More importantly, a man

with a total cholesterol of 200 mg%—a level many feel is risk-free—and an HDL cholesterol of 25 mg% or less may actually have one and a half times the standard risk of developing CHD.

Note: The lab test for HDL cholesterol is simple. The technician adds heparin and manganese chloride to a sample of plasma. This precipitates all lipoproteins except HDL. The precipitates are centrifuged, and an aliquot of the supernatant material, containing only HDL cholesterol, is quantified in the same manner as total cholesterol.

—Castelli

Table 1		
Total cholesterol (mg%)	CHD risk index	Comments
150 or less	*	A level of 150 mg% is near the threshold for CHD; individuals with levels below this rarely have clinical disease. The course of existing lesions probably reverses at 150 mg%.
185	0.667	
200	0.75	Relatively safe level for North Americans.
210	0.80	
220	0.90	Average level of Framingham Study participants who don't develop CHD.
225	1.00	Standard risk.
244	1.50	Average level of Framingham Study participants who get heart attacks.
260	2.00	
300	3.00	

*No numerical value can be given.

Table 2		
HDL cholesterol (mg%)	CHD risk index	
Mg%	Men	Women
75 or more	(Longevity syndrome)	(Longevity syndrome)
70	†	0.52
65	0.45	0.64
60	0.55	0.80
55	0.67	1.00 (standard risk)
50	0.82	1.25
45	1.00 (standard risk)	1.55
40	1.25	1.94
35	1.50	†
30	1.75	†
25 or below	2.00	†

APPENDIX H

INDIVIDUAL DATA FOR GROUP A (HIGH COMPLIANCE, LONG SLOW DISTANCE)

Subject	Age (years)	HDL Cholesterol (mg/dl)		Total Cholesterol (mg/dl)		Maximum Oxygen Uptake (ml/kg/min)		Weight (kg)		Percent body fat		HDL Cholesterol/ Total Cholesterol	
		pre	post	pre	post	pre	post	pre	post	pre	post	pre	post
D.M.	41	33	48	167.0	208.0	45.8	45.8	79.0	77.0	14.0	14.0	.197	.230
J.D.R.	39	64	65	287.0	279.0	45.2	46.2	71.4	71.0	17.0	14.0	.222	.232
T.H.	39	52	68	240.0	225.0	38.2	40.3	91.8	90.0	22.0	22.0	.215	.302
S.R.	42	48	57	191.0	181.0	38.3	51.3	86.0	84.0	19.0	17.0	.251	.314
P.G.	26	54	56	144.0	149.0	32.5	43.5	63.0	62.0	10.0	10.0	.375	.375
M.L.	33	70	85	151.0	173.0	42.8	50.4	61.0	60.0	30.0	24.0	.463	.491
K.P.	26	54	83	202.0	244.0	33.4	49.5	59.0	55.0	28.0	24.0	.267	.340
S.M.	30	52	71	151.0	155.0	30.0	51.2	64.0	63.0	34.0	26.0	.344	.458
C.H.	38	60	64	219.0	220.0	39.2	51.2	55.0	57.0	31.0	26.0	.273	.290
J.DOL.	42	33	52	171.0	208.0	39.8	46.2	93.0	89.0	15.0	16.0	.192	.250
S.S.	42	42	43	216.0	302.0	38.1	45.0	76.0	74.0	12.0	13.0	.194	.142
T.S.	35	49	51	275.0	366.0	44.2	46.5	90.0	87.0	19.0	17.0	.178	.139
F.S.	44	49	67	140.0	192.0	43.5	52.3	81.0	79.0	13.0	12.0	.350	.349
B.L.	36	52	62	215.0	230.0	55.9	55.3	72.0	70.0	10.0	10.0	.242	.269
MEANS	36.6	50.8	62.3	197.8	223.7	40.5	48.2	74.4	72.7	19.6	17.2	.269	.299
CHANGE		11.4		25.9		7.7		-1.7		-2.4		.030	

APPENDIX I

INDIVIDUAL DATA FOR GROUP B (SPORTS)

Subject	Age (years)	HDL Cholesterol (mg/dl)		Total Cholesterol (mg/dl)		Maximum Oxygen Uptake (ml/kg/min)		Weight (kg)		Percent body fat		HDL Cholesterol/ Total Cholesterol	
		pre	post	pre	post	pre	post	pre	post	pre	post	pre	post
J.B.	33	64	57	180.0	157.0	39.5	39.9	84.0	83.0	18.0	13.0	.355	.363
H.J.	28	54	51	185.0	185.0	62.0	81.0	81.0	78.0	11.0	10.0	.291	.275
D.W.	43	64	57	228.0	215.0	34.8	34.8	97.0	95.0	20.0	20.0	.280	.265
C.M.	27	78	66	216.0	217.0	40.2	57.7	75.0	71.0	13.0	10.0	.361	.304
T.V.	30	53	51	281.0	283.0	41.2	41.2	117.0	114.0	14.0	13.0	.188	.180
G.K.	25	51	42	224.0	192.0	34.9	51.0	95.0	93.0	16.0	21.0	.227	.117
MEANS:	31.0	60.6	54.0	219.0	208.2	42.1	50.9	91.5	94.1	15.3	14.0	.284	.251
CHANGE:		-6.7		-10.8		8.8		-2.5		-1.3		-.033	

APPENDIX J

INDIVIDUAL DATA FOR GROUP C (LOW COMPLIANCE, ACTIVE CONTROL)

Subject	Age (years)	HDL Cholesterol (mg/dl)		Total Cholesterol (mg/dl)		Maximum Oxygen Uptake (ml/kg/min)		Weight (kg)		Percent body fat		HDL Cholesterol/ Total Cholesterol	
		pre	post	pre	post	pre	post	pre	post	pre	post	pre	post
E.D.	47	61	54	284.0	263.0	33.5	33.5	102.0	100.0	22.0	13.0	.214	.205
R.S.	23	51	53	197.0	210.0	32.0	29.5	117.0	119.0	25.0	21.0	.258	.252
J.T.	40	66	55	250.0	335.0	32.5	39.0	93.0	91.0	26.0	20.0	.264	.164
J.D.	42	59	55	305.0	294.0	36.7	36.0	80.0	78.0	18.0	17.0	.193	.187
J.D.O.	42	66	62	239.0	218.0	38.9	49.9	91.0	87.0	21.0	14.0	.276	.284
A.B.	38	51	47	227.0	203.0	29.9	43.5	90.0	90.0	19.0	18.0	.224	.231
MEANS:	38.6	59.0	54.3	250.3	253.8	33.9	38.5	95.5	94.1	21.8	17.0	.238	.221
CHANGE:		-4.7		3.5		4.7		-1.4		-4.8		-.018	

APPENDIX K

PRE TEST PROCEDURES

PROJECT LIFESTYLE

Initial Contact Day

1. Blood Pressue
2. Body Fat Measurement
3. Health Risk Analysis and longevity estimate
4. Begin a 12 hour food fast and alcohol abstinence period during the evening
5. Make appointment for Fitness Test

Blood Profile Day

1. Blood profile at Student Health Service as early as possible after 8:00 a.m. (preferably between 8:00-8:30 a.m.).

Fitness Test Day

1. Wear shorts, T-shirt, tennis or jogging shoes.
2. Complete interhealth computerized form.

Tear off and take to Student Health Service
To: Melba Wentz, Lab Tech.

(name)

_____ has been tentatively accepted into Project Lifestyle. A complete blood profile including HDL, will be paid by funds from the Project. Results of the profile should be given to Dr. Bruckner for medical evaluation.

Project coordinators:

Jack Bruckner Ph: 243-2122
Brian Sharkey; Ph: 243-4211
Tom Whiddon: Ph: 243-4211

APPENDIX L

INTERVIEW AND TESTING PROCEDURE

PROJECT LIFESTYLE

Interview and testing procedure.

Day

1. Explain program
2. Show consent form, ask to read, and sign.
3. Inquire as to diet, activity and drugs. Place response on questionnaire.
4. Take Blood Pressure and % Body Fat (calipers and biceps).
5. Administer "Health Risk Analysis and Longevity Estimate."
6. Give participant "Procedure slip" for rest of tests.
7. Explain the need to "fast" for 12 hours preceding test.
8. Collect and take "Health Risk Analysis and Longevity Estimate" to Jack Bruckner at Student Health Service.

APPENDIX M

PROJECT LIFESTYLE QUESTIONNAIRE

EXERCISE:

1. State the exercises in which you are presently participating:
2. How frequently? _____ How long? _____ How vigorously?

3. Other activities
 1. _____ 2. _____
 3. _____ 4. _____
 5. _____
4. Other seasons:

Winter: _____

Spring: _____

Summer: _____

DIET

5. Do you eat pork and/or beef several times a week? _____
How frequently _____
6. How many eggs do you eat per week? _____
7. Do you use butter or margarine? _____
8. Do you use whole milk, or skim milk? _____
9. What type cooking oil is your food cooked in? _____
10. Rate on the scale: High Fat Intake _____ Medium Fat Intake _____
Low Fat Intake _____ Vegetarian _____

DRUGS

11. Do you smoke? _____ How much _____
12. Do you drink alcohol _____ How much _____
13. Do you use drugs for hypertension? What? _____

14. Do you use drugs for depression? What? _____

15. Do you use drugs for anxiety? What? _____

16. Do you use drugs for other medical reasons? What? _____

Comments: _____
